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REVIEW

Evidence of splinting in low back pain? A systematic review of perturbation studies

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Abstract
Purpose The purpose of this systematic review was to assess whether LBP patients demonstrate signs of splinting by evaluating the reactions to unexpected mechanical perturbations in terms of (1) trunk muscle activity, (2) kinetic and (3) kinematic trunk responses and (4) estimated mechanical properties of the trunk.
Methods The literature was systematically reviewed to identify studies that compared responses to mechanical trunk perturbations between LBP patients and healthy controls in terms of muscle activation, kinematics, kinetics, and/or mechanical properties. If more than four studies reported an outcome, the results of these studies were pooled.
Results Nineteen studies were included, of which sixteen reported muscle activation, five kinematic responses, two kinetic responses, and two estimated mechanical trunk properties. We found evidence of a longer response time of muscle activation, which would be in line with splinting behaviour in LBP. No signs of splinting behaviour were found in any of the other outcome measures.
Conclusions We conclude that there is currently no convincing evidence for the presence of splinting behaviour in LBP patients, because we found no indications for splinting in terms of kinetic and kinematic responses to perturbation and derived mechanical properties of the trunk. Consistent evidence on delayed onsets of muscle activation in response to perturbations was found, but this may have other causes than splinting behaviour.

Keywords Low back pain · Perturbations · Trunk · Splinting · Stiffness

Background

It has been suggested that low back pain (LBP) patients splint or guard their lumbar spine through co-contraction of trunk muscles [1]. This could explain observed rigid movement patterns during activities of daily living [2], reduced active range of motion of the lumbar spine [3], the finding that the spinal muscles do not relax in full flexion [4] and increased coupling of pelvis and thorax movements during gait [5, 6]. Splinting could protect the spine from large movement excursions as a result of mechanical perturbations at a cost of an increased axial spinal load, which could negatively affect spine health in the long term [7]. The benefit of splinting through co-contraction is that the concomitant increase in trunk stiffness results in a direct effect, i.e., without delay, on trunk movement when an unexpected external mechanical perturbation is imposed [8]. This would limit the effect of mechanical perturbations on the trunk [9]. Studies on anticipation of- and in

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responses to-trunk perturbations can thus provide evidence for splinting in low back pain patients.

The purpose of this systematic review was to assess whether LBP patients demonstrate signs of splinting, by evaluating the reactions to unexpected mechanical perturbations in terms of (1) trunk muscle activity, (2) kinetic and (3) kinematic trunk responses and (4) estimated mechanical properties of the trunk.

If LBP patients splint their spine, we would expect to find increased trunk muscle activation prior to perturbations. The resulting increased initial resistance to the perturbation should increase initial kinetic responses when perturbations are position-controlled or decrease the amplitude and rate of change of trunk kinematics when perturbations are force-controlled. Both would be reflected in higher estimates of trunk stiffness. Slower trunk movements after force-controlled perturbations would most likely result in a later detection of movement by the sensory system and consequently to a later onset of reactive muscle activation.

Different muscle recruitment patterns to stabilize the lumbar spine have been suggested to be present between subjects in the LBP population [10, 11], which would result in a higher between subject variance among LBP patients than among controls. Since this may mask group differences when summary statistics are presented, the between subject variance of outcomes was also evaluated.

Methods

Search strategy

The literature was systematically reviewed to identify studies that compared the response to mechanical trunk perturbations between LBP patients and healthy controls. The search strategy contained five blocks: (1) low back pain, (2) perturbations, (3) muscular response, (4) kine(ma)tic response and (5) estimated mechanical trunk properties. Titles, abstracts or keywords had to contain strings from both first two blocks and at least one from blocks three to five. The search is outlined in supplement 1.

In July 2015, the systematic search was performed in the following databases: Academic Search Premier, CINAHL, EMBASE, MEDLINE, and ScienceDirect. No limits were set for study design or publication date. First, all titles were screened for relevance by the first (MP) and second (MG) author. Both selections of possibly relevant studies were combined. The selection of abstracts was performed in the same manner. Studies were in-or excluded by screening of the selected full-texts using the criteria presented below. Differences in judgement were resolved during a consensus procedure in which the first two authors discussed these papers until agreement about inclusion was reached.

Inclusion and exclusion criteria

Studies had to use experimental setups in which unexpected mechanical perturbations were imposed to subjects with LBP and to healthy controls. The effect of the perturbations on the trunk had to be reported in at least one of the four following terms: (1) muscular response (2) kinetic response, (3) kinematic response (4) estimated mechanical trunk properties. A quantitative or statistical comparison between LBP patients and healthy controls had to be presented. If subjects could anticipate some of the imposed perturbations a separate analysis of the reactions to unexpected perturbations had to be presented. Studies that experimentally induced LBP in healthy controls were excluded. There were no restrictions on duration or diagnosis (non-specific or specific) of LBP.

Data extraction

Data extracted by the first author (MP) consisted of subject characteristics, experimental set-up, normalization procedures, and differences in reported outcomes between control subjects and LBP patients expressed as means, variances and levels of statistical significance.

Pooling of results was performed, first, to pinpoint common patterns specific to LBP patients vs. controls. Outcomes were assigned to one of nine blocks: pre-perturbation muscle activity, timing and amplitude of muscle, kinetic and kinematic responses, and estimated trunk stiffness and damping. If three or more studies reported the statistical significance of between group differences in a block, pooling of results within that block was performed. The average percentage of significantly higher (or lower) values in the LBP group within that block was calculated for each study and then averaged over studies. For each block we considered the evidence for splinting behaviour in LBP to merit further attention if the average percentage of outcomes that were significantly higher (or lower) in LBP patients was 40% or more. The methods and results of pooling of variances are outlined in supplement 3.

Results

Systematic search

The search yielded a total of 571 studies. After reviewing titles and abstracts, 36 studies remained that were subjected to a full-paper review. Screening of the reference lists yielded no extra studies. Ultimately, 19 studies were

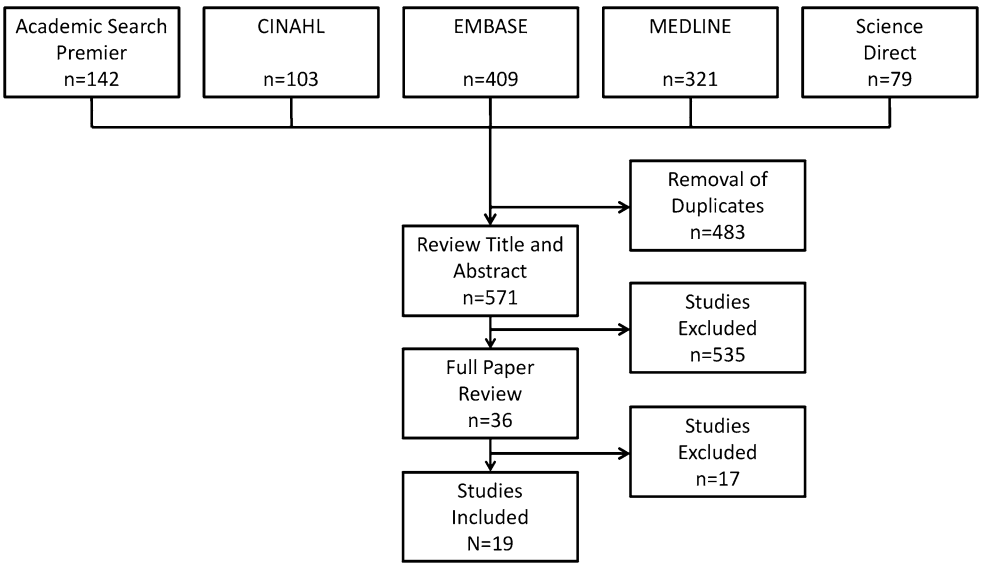

Fig. 1 Flowchart of the selection process

included in this review. A flow-chart is presented in Fig. 1. A library (Endnote, Thomson Reuters, New York) containing the evaluated titles and abstracts of the selection procedure is presented in supplement 2.

Data extraction

Subject characteristics

An overview of subject characteristics is presented in Table 1. The 19 included studies contain the results of 17 unique cohorts [12–30], consisting of 286 LBP patients and 306 healthy controls. Two cohorts were presented twice ([12, 25] and [15, 24]). The mean age of participants was between 20 and 45 years. LBP patients generally had higher body mass (14 out of 18 studies) and Body Mass Indices than healthy controls (6 out of 7 studies), although none of the studies reported these between group differences to be significant. Twelve studies included LBP patients that had experienced pain for 3 months or more. LBP intensity was assessed using a Visual Analogue Score or a Numeric Rating Scale and the mean value in LBP subjects varied from 1.7 to 6.1 out of 10. One study measured patients with disc herniation that were selected for micro-discectomy because of prolonged LBP with sciatica [15, 24]. The other studies included patients with non-specific LBP.

Experimental setup

An overview of the experimental setups is presented in Table 2. In all experiments, subjects held the trunk in an upright position before being perturbed. Perturbations were imposed in a standing position in 11 studies [14–17, 20, 22, 24, 26–28, 30], semi-seated, i.e., with the hips bent 45° and knees in 90°, in five [13, 18, 19, 23, 29] and seated in three [12, 21, 25] (Fig. 2). In seven studies, the perturbations were imposed directly to the trunk [13, 18–21, 23, 29]. In only one of these experiments the perturbation was position controlled [21], the other studies imposed [20, 23, 29] or released [13, 18, 19] a force. In the other experiments the perturbations were imposed indirectly to the trunk, either via the arms [14–16, 22, 24, 26, 28] or the legs [12, 17, 25, 27, 30] (Fig. 3).

In 13 studies, the pelvis of participants was fixated during the experiment [12–15, 18–25, 29]. In two of these studies (describing one cohort), the lower extremities were fixated to a 'swing chair' that could tilt around a medio-lateral axis allowing movement in the sagittal plane. This chair was tilted backward to a fixed angle and then released. Subjects were instructed to regain a balanced upright position [12, 25]. In the six studies in which the pelvis was not fixated, three imposed horizontal translations of the standing surface [17, 27, 30] and three perturbed the trunk via the arms, either by pulling one arm downward [16, 26] or by dropping a weight in a box held by the participant [28]. Muscular activation was evaluated in 15 studies [12–20, 22–24, 27, 29, 30], the kinetic response in two studies [12, 30], and the kinematic response in five studies [12, 21, 25, 26, 28]. Mechanical trunk properties were estimated in two studies [21, 25].

Muscle activation

An overview of the studies assessing muscle activation is presented in Table 3. Of these sixteen studies, five

Table 1 Subject characteristics

Studies	Group	*n* (*m*/*f*)	Age	Height in cm	Weight in kg	BMI in kg/m^2	Pain score type	LBP pain score	Definition LBP
[12, 25][c1]	LBP	23 (11/12)	36.8 (11.6)	169 (8.4)	69 (12)	24.1 (3.1)	VAS for severity of pain	3.8 (1.0)	Subacute; >6 weeks
	Control	31 (16/15)	31.7 (8.1)	168 (9.8)	64 (13)	22.5 (2.7)		NA	
[26]	LBP	10 (0/10)	40.6 (11.6)	NR	62 (7)	NR	VAS current LBP	2.9 (2.1)	Minimum duration of 2 years
							VAS LBP over the last 4 weeks	3.5 (1.4)	
	Control	10 (0/10)	39.7 (14)	NR	62 (8)	NR		NA	
[30]	LBP	16 (8/8)	33.9 (6.2)	175 (9)	74 (9)	24.3 (2.6)	NRS pain	3 (range 0–7)	Severe enough to seek treatment
	Control	16 (8/8)	33.5 (9.0)	171 (7)	69 (12)	23.4 (3.2)		NA	
[23]	LBP	27 (14/13)	*m* = 43 (10)	*m* = 174 (6)	*m* = 78 (13)	*m* = 26 (4)	VAS pain intensity	m = 3.7 (2.2)	Daily or almost daily pain for at least 3 months
			f = 35 (9)	*f* = 163 (6)	*f* = 69 (14)	*f* = 26 (5)		f = 3.4 (2.7)	
	Control	29 (15/14)	*m* = 38 (10)	m = 174 (7)	*m* = 78 (9)	*m* = 26 (3)		NA	
			f = 39 (10)	*f* = 165 (9)	*f* = 63 (7)	*f* = 23 (3)			
[15][c2]	LBP	20 (15/5)	39 (10)	175 (7)	78 (16)	NR	VAS back pain intensity	6.1 (1.9)	Disc herniation selected for microdiscectomy as a result of prolonged LBP with sciatica
	Control	15 (10/5)	37 (12)	175 (9)	74 (13)	NR		NA	
[24][c2]	LBP	16 (NR/NR)	NR	NR	NR	NR	↩	5.2 (range 0.9–8.5)	↩
	Control	↩	↩	↩	↩	↩		NA	
[16]	LBP	17 (0/17)	40.9 (11.9)	167 (6)	62 (9)	22.1 (2.5)	NRS Current pain intensity before perturbation	3.5 (2.5)	Minimum duration of 6 months
	Control	17 (0/17)	34.0 (11.3)	168 (6)	59 (8)	20.9 (2.7)		NA	
[21]	LBP	8 (8/0)	20.4 (1.6)	183 (5)	73 (3)	NR	VAS LBP	2.5 (0.9)	Recurrent acute exercise induced LBP for at least 6 months
	Control	8 (8/0)	20.7 (1.0)	179 (3)	71 (7)	NR		NA	
[28]	LBP	11 (NR/NR)	28.5 (5.8)	175 (13)	74 (14)	24.0 (2.5)	VAS level of pain just before testing	1.7 (1.9)	Sick leave from usual occupation or treatment, which lasted for more than 18 months with at least one episode of LBP in the preceding 6 months or pain that was semi continuous with periods of greater and lesser pain
	Control	11 (NR/NR)	27.5 (4.3)	175 (6)	71 (13)	23.1 (3.4)		NA	
[29]	LBP	13 (7/6)	32.3 (8.2)	175 (8)	81 (20)	26.4 (6.0)	NRS mean score of (1) current pain intensity and (2) best and (3) worst pain over last 24 h	3.6 (1.3)	Duration of pain less than or equal to 8 weeks, experienced at least one separate episode in the past year that had resolved
	Control	13 (7/6)	35 (10.1)	175 (12)	85 (20)	27.3 (4.2)		NA	
[17]	LBP	20 (9/11)	37 (10.1)	171 (11)	78 (16)	NR	VAS pain on day of testing	4.7 (range 2.1–7.7)	Pain between L1 and gluteal folds for at least 6 months
	Control	20 (9/11)	37 (9.6)	171 (9)	74 (14)	NR		NA	
[27]	LBP	8 (0/8)	42.4 (14.5)	168 (4)	65 (9)	23.1 (2.4)	VAS pain intensity after perturbations	5.2 (3.5)	At least 6 months several times a week or daily back pain
	Control	12 (0/12)	27.3 (7.1)	168 (6)	57 (6)	20.4 (2.6)		NA	

Table 1 continued

Studies	Group	*n* (*m*/*f*)	Age	Height in cm	Weight in kg	BMI in kg/m^2	Pain score type	LBP pain score	Definition LBP
[18]	LBP	17 (12/5)	*m* = 35.1 (12.4) *f* = 43.8 (7.5)	NR	*m* = 84 (15) *f* = 68 (12)	NR	VAS extent of pain	NR	Periodic back pain episodes for more than 6 months
	Control	17 (12/5)	*m* = 35.9(12.6) *f* = 45(9.8)	NR	*m* = 78 (18) *f* = 59 (13)	NR		NA	
[13]	LBP	16 (15/1)	38.8 (10.1)	176 (9)	82 (15)	NR	VAS overall back pain	2.7 (2.0)	LBP for periods ranging from 6 months to 35 years
	Control	14 (13/1)	38.1 (9.6)	177 (9)	80 (18)	NR		NA	
[22]	LBP	25 (18/7)	40.7 (10.6)	174 (9)	73 (8)	NR	VAS back pain	2.7 (2.4)	Chronic or non-specific mechanical recurrent LBP having a pain history of 3 months, without radiation
	Control	25 (15/10)	32.2 (9.6)	172 (12)	72 (9)	NR		NA	
[19]	LBP	20 (4/16)	*m* = 37.3 (10.6) *f* = 35.8 (9.0)	*m* = 177 (10) *f* = 164 (7)	*m* = 81 (14) *f* = 65 (10)	NR	NR	NR	Experienced back pain for at least 6 months
	Control	20 (4/16)	*m* = 38.7 (12.1) *f* = 34.5 (12.8)	*m* = 179 (7) *f* = 164 (10)	*m* = 81 (17) *f* = 61 (14)	NR		NA	
[14]	LBP	24 (16/8)	24.3 (4.7)	174 (11)	70 (8)	NR	VAS pain	3.2 (1.6)	LBP for at least 3 months
	Control	25 (17/8)	25.1 (5.1)	172 (10)	67 (8)	NR		NA	
[20]	LBP	21 (11/10)	*m* = 28.4 (8.4) *f* = 34.2 (10.4)	*m* = 179 (4) *f* = 163 (8)	*m* = 77 (10) *f* = 68 (11)	NR	VAS LBP	NR	Episodic LBP, VAS for LBP greater than 3/10 on day of testing
	Control	23 (15/8)	*m* = 30.3 (9.1) *f* = 33.5 (13.2)	*m* = 168 (4.6) *f* = 163 (6)	*m* = 82 (14) *f* = 61 (11)	NR		NA	

Parenthesized values are standard deviations, unless stated otherwise

cn the subjects described in these studies belong to the same cohort, *f* female, *LBP* low back pain, *m* male, *NR* not reported, *NRS* numeric rating scale, *NA* not applicable, *VAS* visual analog scale, ⟷ the same value/content as above

Table 2 Experimental setups

Studies	Subject position	Fixed segments	Perturbation	Conditions	n-Perturb. per condition	Subject instruction	Reported outcomes
[12][c1]	Seated	Pelvis, lower limbs	Swingchair release from a 20° backwards tilted position	NA	1	Achieve balanced position	EMG, kinematics, kinetics
[25][c1]	Seated	Pelvis, lower limbs	Swingchair release from a backwards tilted position	10° and 20° backward tilted starting position	3	Achieve balanced position	Kinematics, mechanical trunk properties
[26]	Standing	None	Unilateral downward pull handheld grip	Left arm/right arm	5	Stay erect, look straight ahead	Kinematics
[30]	Standing	None	Horizontal position controlled 10 cm translation of floor	12 perturbation directions (1–12 o'clock)	4	Stand comfortably, look forward	EMG, kinetics
[23]	Semi-seated	Pelvis, lower limbs	Continuous forward pull at T4 level with unexpected additional load	NA	20	NR	EMG
[15][c2]	Standing	Pelvis, T6	Weight dropped in box held by participant	(1) Unexpected (eyes open)/expected (eyes closed), (2) Supported/unsupported stance	3	NR	EMG
[24][c2]	Standing	Pelvis, T6	Weight dropped in box held by participant	(1) Unexpected (eyes open)/expected (eyes closed), (2) Supported/unsupported stance	3	NR	EMG
[16]	Standing	None	Force controlled unilateral downward pull handheld grip of 150 N (built up in 100 ms), left hand	NA	5	NR	EMG
[21]	Seated	Pelvis	Position controlled horizontal push 10 mm in ~40 ms at T8 level	Anterior push/posterior push	12	Sit upright, do not resist or intervene with perturbations	Kinematics, kinetics, mechanical trunk properties
[28]	Standing	None	Drop 1 kg weight from 30 cm height in container held in hands with 90° elbow flexion	Standing of flat surface or short base (block of 12 cm antero–posterior length)	5	Maintain equal weight bearing, maintain positions of the elbows during perturbation	Kinematics
[29]	Semi-seated	Pelvis, lower limbs	Continuous 100 N anterior pull at T6–T7 level with force controlled pseudo-random perturbations of additional load	Additional load of + or − 30 N	±36	NR	EMG
[17]	Standing	None	Horizontal position controlled translation or rotation of surface	(1) Forward/backward/rotation (2) Medium/large (only for translations)	16	NR	EMG

Table 2 continued

Studies	Subject position	Fixed segments	Perturbation	Conditions	n-Perturb. per condition	Subject instruction	Reported outcomes
[27]	Standing	None	Lateral perturbation of surface of approximately 3 cm	Eyes open/closed	7	NR	EMG
[18]	Semi-seated	Pelvis, lower limbs	Sudden load release at T9 level	(1) Forward/backward (2) Left/right (3) 20%/30% of maximal isometric trunk exertion	3	NR	EMG
[13]	Semi-seated	Pelvis, lower limbs	Sudden load release at T9 level	(1) Forward/backward (2) Left/right (3) 20%/30% of maximal isometric trunk exertion	3	NR	EMG
[22]	Standing	Pelvis	Drop of 3 kg steel cylinders onto the outstretched hand	(1) Expected/unexpected (2) Solid/foam surface	3	Do not resist before the impact or let go along with the perturbation after the impact. Return to previous position as quickly as possible	EMG
[19]	Semi-seated	Pelvis, lower limbs	Load release at T9 level of 40 N (females) or 65 N (males)	Flexion/extension/lateral bending	3	Keep force output line steady at target and do not anticipate release	EMG
[14]	Standing	Pelvis	Drop of 3 kg weight into outstretched hand from 8 cm height	Expected/unexpected (blindfolded)	3	Let go, do not resist the perturbation	EMG
[20]	Standing	Pelvis	Force controlled superimposed full sinewave (80 ms) on top of horizontal preload at level of T12	(1) Pull direction 0°/45°/90°/135°/180° to anterior direction (2) Preload 15/30% of maximal effort (3) Superimposed perturbation of 5/10% of max. effort	3	Maintain the target effort until after the perturbation	EMG

cn = The subjects described in these studies belong to the same cohort, *EMG* electromyography, *NA* not applicable, *NR* not reported, ⟷ the same value/content as above

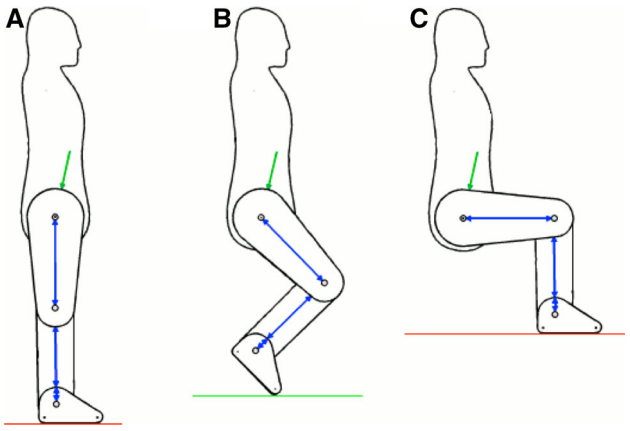

Fig. 2 Body Positions. Perturbations were imposed to subjects that were in a standing (**a**), semi-seated (**b**), or seated (**c**) position. The images show e-Verne from wwrichard.net, with permission

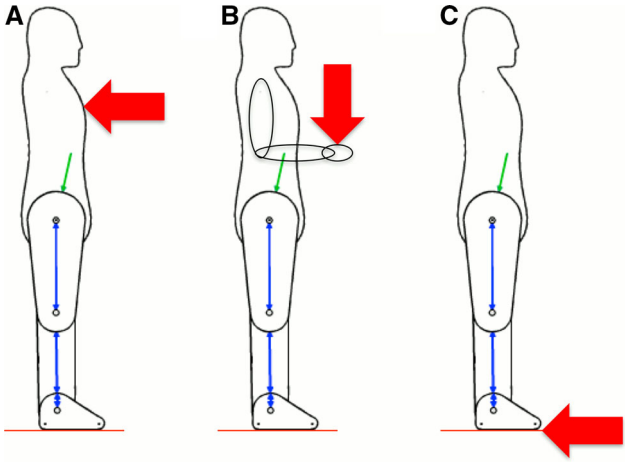

Fig. 3 Trunk Perturbation Types. Trunk perturbations were imposed directly to the trunk (**a**), or indirectly, either via the arms (**b**) or legs (**c**). *Red arrows* indicate the locus of the perturbation; the direction varies within and between studies. The images show e-Verne from wwrichard.net, with permission

evaluated the pre-perturbation activity of trunk muscles [16, 21, 23, 27, 30]. In one study [23], a significantly higher pre-activation of several back muscles was reported in LBP patients, both after normalization to a reference contraction and to a maximal voluntary contraction (MVC). In one study that normalized to the maximal amplitude of each muscle measured over the entire experiment, a significantly lower pre-activation of one abdominal muscle was reported [30]. In the other three studies, that either used no normalization [16, 27] or MVC normalized EMG [21], no significant between group differences were reported for pre-activation of abdominal or back muscles.

Eleven studies evaluated the response time of trunk muscle activation, i.e., the time between the perturbation and the first muscular response [12–16, 18, 21, 23, 24, 27, 29]. In eight of these studies, the first muscular response was defined as the instant at which an EMG signal exceeded a predetermined number of standard deviations above baseline activity, varying from 1.4 to 3 standard deviations [12–14, 16, 18, 21, 23, 29]. Six of these studies reported significantly longer response times in multiple trunk muscles [13, 14, 16, 18, 21, 29]. A significantly shorter response time in LBP trunk muscles was reported in the experiment in which a swing-chair was used [12]. One study, additionally used an approximated generalized likelihood-ratio (AGLR) method to estimate response times [23]. Neither method showed a significant between group difference. Two studies on one cohort found no between-group differences on visually detected response times [15, 24]. One study did not report how the response time was determined and found no significant between group differences [27].

The amplitude of trunk muscle activation in response to perturbations was assessed in six studies [16, 22, 23, 27, 29, 30]. Of the three studies that did not normalize the EMG signals of back and abdominal muscles [16, 22, 27], two reported no between group differences [16, 27]. One study found that the maximal amplitude of LBP patients' trunk muscles was lower over a time window of 40–120 ms after perturbation, but higher if this window was increased to 40–250 ms after perturbation. One study normalized by dividing the linear EMG envelope by the maximum value measured over all perturbations for that specific muscle, and found higher activation of both abdominal and back muscles in LBP patients [30]. Higher amplitudes of back muscle activation were also found in another study using either no normalization or a normalization to a reference contraction [23]. One study reported the opposite, i.e., lower back muscle EMG amplitudes normalized to a reference contraction in LBP patients [29].

Kinematic response to perturbations

An overview of the five studies that assessed kinematic outcomes is presented in Table 4. Two studies imposed a backwards tilt followed by release of a swing chair in one cohort of subjects [12, 25]. These studies reported larger sagittal plane angular velocity of the hip in LBP patients, but not of the lumbar spine. In patients, the sagittal range of motion (defined as the maximum minus the minimum angle measured from chair release until the time a balanced position was achieved) was significantly smaller for the lumbar spine but larger for the hip. It took subjects between 4 and 5 s to regain balance with no significant group difference. One study assessed the effect of a downward arm pull on trunk kinematics [26]. This study reported that subjects with LBP showed a smaller caudal movement of both posterior superior iliac spines (PSIS) and a greater

Table 3 Muscle activation

Studies	EMG parameter	Amplitude normalization/threshold definition	Time window	Perturbation	Muscle	LBP outcome	Control outcome	*p* value pain status
[12][c1]	Relative duration of co-contraction of all studied trunk muscles (%)	3 SD above baseline activity	Chair release—'balance achieved'	20° tilt	RA, EO, IO, ES	35.2	12.6	<0.05
	Relative duration of muscle contraction (%)	↩	↩	↩	↩	Each muscle increased[$]	Each muscle decreased[$]	Each muscle <0.05
	Response time (ms)	↩	Chair release—muscle contraction	↩	↩	Each muscle decreased[$]	Each muscle increased[$]	Each muscle <0.05
	Left–right muscle symmetry (cross-correlation)	NA	Chair release—'balance achieved'	↩	***RA***,	0.61 (0.13)	0.70 (0.11)	NS
					EO,	0.74 (0.18)	0.75 (0.21)	NS
					IO,	0.69 (0.24)	0.62 (0.15)	NS
					ES	0.56 (0.15)	0.67 (0.09)	<0.05
[30]	Pre-activation amplitude (dimensionless)	Maximal value of that muscle measured over all perturbation directions	250–50 ms before perturbation	NA	***REO***	Decreased[$]	Increased[$]	0.044
	Response amplitude (dimensionless)*	↩	**100–175 ms after perturbation**	All directions combined	***LEO***	Increased[$]	Decreased[$]	0.019
			25–100 ms after perturbation		***LES3***	Increased[$]	Decreased[$]	0.030
			100–175 ms after perturbation		***LES3***	Increased[$]	Decreased[$]	0.0062
[23]	Pre-activation amplitude (dimensionless)	Reference contraction	250–0 ms before perturbation	NA	***L5***	m 20 (IQR 15–27)	m 18 (IQR 16–19)	0.038
						f 23 (IQR 14–30)	f 17 (IQR 6–19)	
					L3	m 15 (IQR 12–21)	m 11 (IQR 9–14)	0.017
						f 24 (IQR 15–27)	f 15 (IQR 6–21)	
					L1	m 24 (IQR 14–31)	m 20 (IQR 17–23)	NS
						f 22 (IQR 15–31)	f 17 (IQR7–25)	
					T10	m 25 (IQR 18–33)	m 18 (IQR 11–22)	NS
						f 18 (IQR 12–51)	f 21 (IQR 9–30)	
					RA	m 20 (IQR 10–28)	m 18 (IQR 10–39)	NS
						f 24 (IQR 11–35)	f 31 (IQR 15–35)	
					EO	m 15 (IQR 8–28)	m 19 (IQR 10–34)	NS
						f 28 (IQR 18–48)	f 24 (IQR 16–36)	

Table 3 continued

Studies	EMG parameter	Amplitude normalization/threshold definition	Time window	Perturbation	Muscle	LBP outcome	Control outcome	*p* value pain status
	↩	Maximal voluntary contraction	↩	↩	***L5***	m 10 (IQR 6–21) f 11 (IQR 6–20)	m 6 (IQR 5–8) f 8 (IQR 6–12)	0.042
					L3	m 5 (IQR 2–8) f 4 (IQR 3–9)	m 3 (IQR 2–4) f 4 (IQR 2–5)	0.019
					L1	m 11 (IQR 4–19) f 8 (IQR 7–13)	m 5 (IQR 4–7) f 7 (IQR 4–9)	0.009
					T10	m 6 (IQR 4–12) f 5 (IQR 4–12)	m 4 (IQR 3–6) f 5 (IQR 4–7)	NS
	Response time (ms)	'SD' {Hodges [41] #1119} and 'AGLR' {Staude [42] #1122} method	Perturbation onset—muscle 'on'	Forward pull T4	L5, L3, L1, T10	All NR	All NR	All NS
	Amplitude first EMG peak (dimensionless)	Muscle activity 250 ms prior to perturbation	30–150 ms after perturbation	↩	***L5, L1, T10***	All NR	All NR	All NS
					L3	Increased[$]	Decreased[$]	0.025
	Amplitude first EMG peak (μVolt)	None	↩	↩	***L5, L1, T10***	All NR	All NR	All NS
					L3	Increased[$]	Decreased[$]	0.008
[15][c2]	Response time (ms)	Visual inspection rectified signal	Perturbation onset—muscle 'on'	Weight dropped in box in hands		***Supported stance***	***Supported stance***	
					T12	47.7 (15)	57.2 (36)	NS[¥]
					L5	42.8 (9)	41.5 (14)	NS[¥]
						Unsupported	***Unsupported***	
					T12	43.0 (17)	41.0 (20)	NS[¥]
					L5	41.9 (11)	34.9 (11)	NS[¥]
[24][c2]	↩	↩	↩	↩	↩	NR	NR	NS[¥]
[16]	Preactivation amplitude (μVolt)	None	300–0 ms before perturbation	NA	***RA***	4 (3)	2 (1)	NS
					OE	9 (8)	6 (3)	NS
					OI	14 (13)	14 (11)	NS
					ES	2 (2)	3 (2)	NS
					MF	3 (3)	2 (2)	NS
	Response time (ms)	4 SD above baseline activity	0–200 ms after perturbation	Left hand downward pull	***RA***	54 (11)	39 (11)	<0.01
					OE	39 (11)	33 (6)	NS
					OI	76 (26)	55 (11)	<0.01
					ES	74 (39)	57 (31)	NS
					MF	85 (37)	79 (43)	NS
	Response amplitude (μVolt)	None	↩	↩	***RA***	42 (36)	65 (53)	NS
					OE	88 (53)	104 (41)	NS
					OI	79 (71)	85 (43)	NS
					ES	26 (24)	30 (29)	NS
					MF	27 (25)	29 (34)	NS

Table 3 continued

Studies	EMG parameter	Amplitude normalization/threshold definition	Time window	Perturbation	Muscle	LBP outcome	Control outcome	*p* value pain status
[21]	Pre-activation amplitude (dimensionless)	MVC	400–0 ms prior to perturbation onset	NA	L3	NR	NR	NS
	Response time (ms)	2 SD above baseline activity	Perturbation onset—muscle 'on'	10 mm anterior trunk push in ~40 ms	↩	Increased[$]	Decreased[$]	0.006
[29]	Response time (ms)	2 SD above baseline activity	Perturbation onset—muscle 'on'	Continuous 100 N anterior pull at T6–T7 level with pseudorandom perturbations of additional load	***MF(fw)***	44 (9.5)	36.9 (6.8)	0.014
					MF	42.7 (3.0)	37.3 (5.1)	0.016
					ES	43.1 (4.7)	36.4 (5.2)	0.001
	Response amplitude (% Sorenson)	Biering-Sorenson test {Biering-Sorensen, 1984 #1121}	↩	↩	***MF(fw)***	6.1 (4.1)	9.1 (5.1)	0.030
					MF	7.1 (3.9)	9.7 (4.1)	NS
					ES	5.3 (3.8)	8.6 (4.4)	NS
[17]	Subjects in whom muscle firing was detected on at least 1 side (%)	Above 99% CI baseline activity	0-750 ms after perturbation onset	Backward large	***ES***	85	100	<0.05
				Backward medium	***RA***	65	75	<0.05
				Forward large	***ES***	75	90	<0.05
				Forward medium	***RA***	50	55	<0.05
				Toes up	***ES***	80	90	<0.05
					RA	90	90	NS
					ES	65	70	<0.05
					RA	70	70	<0.05
					ES	75	75	<0.05
					RA	15	50	<0.05
[27]	Preactivation amplitude (μVolt)	None	NR	NA	RA, OI, OE, MF, ES	All NR	All NR	All NS
	Response time (ms)	Detected by matlab and visually corrected	↩	Mediolateral	↩	All NR	All NR	All NS
	Response amplitude (AUC)	Detected by matlab and visually corrected	↩	↩	↩	All NR	All NR	All NS
	Response amplitude (μVolt)	None	↩	↩	↩	All NR	All NR	All NS
[18]	Mean number of antagonists 'on' (*n*)	1.4 SD above baseline activity for at least 25 ms	NR	Sudden release pull towards	RA EO, IO, LD, T9, L3			
				Extension		5.4 (1.1)	5.8 (0.5)	<0.01
				Flexion		5.4 (0.8)	5.3 (0.8)	NS
				Lateroflexion		5.2 (1.0)	5.7 (0.4)	<0.01
	Mean number of agonists 'off' (*n*)	1.4 SD below baseline activity for at least 44 ms	↩	***Extension***	↩	4.7 (1.1)	5.1 (1.1)	<0.01
				Flexion		3.0 (2.9)	4.3 (2.1)	<0.01
				Lateroflexion		2.5 (1.3)	3.7 (1.1)	<0.01
	First antagonist muscle 'on' (ms)	1.4 SD above baseline activity for at least 25 ms	↩	***Extension***	↩	63 (18)	56 (11)	<0.01
				Flexion		59 (14)	53 (10)	NS
				Lateroflexion		61 (11)	54 (6)	<0.01
	First agonist muscle 'off' (ms)	1.4 SD below baseline activity for at least 44 ms	↩	***Extension***	↩	35 (12)	30 (8)	<0.01
				Flexion		42 (27)	32 (15)	<0.01
				Lateroflexion		50 (52)	30 (10)	<0.01

Table 3 continued

Studies	EMG parameter	Amplitude normalization/threshold definition	Time window	Perturbation	Muscle	LBP outcome	Control outcome	*p* value pain status
	Mean antagonistic muscle reaction time (ms)	1.4 SD above baseline activity for at least 25 ms	⟷	***Extension***	⟷	87 (34)	69 (18)	<0.01
				Flexion		85 (25)	69 (8)	<0.01
				Lateroflexion		86 (16)	74 (15)	<0.01
	Mean agonistic muscle reaction time (ms)	1.4 SD below baseline activity for at least 44 ms	⟷	***Extension***	⟷	72 (32)	57 (21)	<0.01
				Flexion		92 (70)	60 (29)	<0.01
				Lateroflexion		83 (55)	55 (23)	<0.01
[13]	Mean antagonistic muscle respone time ON (ms)	1.4 SD above baseline activity for at least 25 ms	0-300 ms after perturbation onset	Sudden release pull towards	RA EO, IO, LD, T9, L3			
				Extension		74 (15)	69 (18)	<0.05
				Flexion		80 (20)	63 (9)	<0.05
				Lateroflexion		80 (16)	70 (13)	<0.05
	Agonistic muscle response time OFF (ms)	1.4 SD below baseline activity for at least 44 ms	⟷	***Extension***	⟷	63 (27)	53 (37)	<0.05
				Flexion		68 (40)	68 (40)	<0.05
				Lateroflexion		57 (21)	53 (20)	NS
[22]	Response amplitude (dimensionless)	None	40 ms to 120 ms after perturbation onset	Sudden release of 3 kg steel cylinders onto the outstretched hand	***RA***	Decreased[$]	Increased[$]	0.05
					ES	Decreased[$]	Increased[$]	0.02
	⟷	⟷	40 ms to 250 ms after perturbation onset	⟷	***RA***	Increased[$]	Decreased[$]	0.05
					ES	Increased[$]	Decreased[$]	0.05
[19]	Mean antagonistic muscle response time ON (ms)	1.5 SD above baseline activity	0–2 s after perturbation onset	Sudden release pull towards	RA EO, IO, LD, T9, L5			
				Extension		87 (28)	74 (20)	NS
				Flexion		82 (15)	62 (10)	<0.001
				Left		87 (15)	78 (27)	NS
				Right		91 (32)	74 (15)	0.044
	Mean antagonistic muscle response time OFF (ms)	1.5 SD below baseline activity	⟷	***Extension***	⟷	65 (18)	44 (11)	<0.001
				Flexion		99 (67)	58 (37)	0.028
				Left		81 (38)	54 (28)	0.019
				Right		62 (41)	45 (22)	NS
[20]	Muscles activated in response to perturbation (%)	Shewhart method {Hodges, 1996 #1119}	25–150 ms after perturbation onset	Trunk pull in multiple directions	⟷	3.5	4.3	NS
	Mean difference pre- and post activation amplitudes (dimensionless)	MEMGD method {Stokes, 2000 #1120}	25–150 ms before and after perturbation onset	⟷	⟷	0.545	0.548	NS

Parenthesized values are standard deviations, unless stated otherwise. Bold italic words are linked to presented mean values and levels of significance on the same horizontal level

AUC area under the curve, *CI* confidence interval, *cn* the subjects described in these studies belong to the same cohort, *ES* m. erector spinae, *f* female, *fw* finewire EMG, *IQR* interquartile range (Q1–Q3), LD, m. latissimus dorsi, *Ln* lumbar erector spinae at level of *n*th vertebrae, *m* male, *MEMGD* mean EMG difference, *MF* m. multifidus, *MVC* maximally voluntary contraction, *NS* not significant, *OI* m. obliquus internus, *OE* m. obliquus externus, *Tn* thoracic erector spinae at level of *n*th vertebrae, ⟷ the same value/content as above

*Only significant results are reported

[$] No mean values reported

[¥] Statistical test was performed with results of expected and unexpected perturbations combined

Table 4 Kinematic response to perturbations

Study	Kinematic parameter	Time window/instant	Perturbation	Joint	LBP outcome	Control outcome	*p* value pain status
[12]	RMS angular velocity (rad/s)	Chair release to 'balance achieved'	20° tilt	***Hip***	0.44 (0.11)	0.35 (0.11)	<0.05
				Lumbar spine	0.13 (0.07)	0.16 (0.09)	NS
	Maximum—minimum angular velocity (rad/s)	↩	↩	***Hip***	1.91 (0.56)	1.50 (0.53)	<0.05
				Lumbar spine	0.58 (0.34)	0.71 (0.36)	NS
[25]	Maximum—minimum angle (deg)	Chair release to 'balance achieved'	10° tilt	***Hip***	26.6 (9.3)	20.6 (6.9)	<0.05
				Lumbar spine	7.9 (3.5)	9.9 (5.3)	<0.05
				Chair	22.4 (7.2)	22.4 (6.1)	NS
	↩	↩	20° tilt	***Hip***	31.8 (8.6)	25.3 (9.1)	<0.05
				Lumbar spine	9.2 (5.3)	13.3 (7.6)	<0.05
				Chair	27.5 (6.0)	28.4 (6.3)	NS
	Spine/hip angle ratio (dimensionless)	At first peak angle chair	***10° tilt***	Hip and lumbar spine	0.24 (0.16)	0.47 (0.39)	NS
			20° tilt		0.26 (0.29)	0.58 (0.67)	NS
	↩	At second peak angle chair	***10° tilt***	↩	0.18 (0.14)	0.41 (0.35)	NS
			20° tilt		0.31 (0.32)	0.53 (0.63)	NS
	Balancing error (deg)	At balanced position	***10° tilt***	Chair	2.6 (2.5)	1.8 (1.6)	NS
			20° tilt		2.3 (1.7)	1.9 (2.0)	NS
	Time to regain balance (s)	Chair release to 'balance achieved'	***10° tilt***	↩	4.5 (2.1)	4.3 (1.4)	NS
			20° tilt		4.3 (1.6)	4.6 (2.0)	NS
[26]	***MAPCH (mm)***	Perturbation onset to 1 s after	Arm pull	NA	13.0 (5.0)	8.4 (4.4)	0.04
	MAPISIPS (mm)				7.6 (3.8)	3.3 (3.4)	0.02
	MCMCSIPS (mm)				6.1 (2.1)	8.5 (4.0)	NS
	MCMISIPS (mm)				5.6 (2.4)	8.5 (4.0)	NS

Table 4 continued

Study	Kinematic parameter	Time window/instant	Perturbation	Joint	LBP outcome	Control outcome	*p* value pain status
[21]	Peak anterior trunk velocity (mm/s)	NR	10 mm anterior trunk push in ~40 ms	NA	NR	NR	NS
[28]	Onset anterior translation of L1 (ms)	NR	Drop 1 kg weight in hands on	NA			
			Flat surface		163.7 (38.3)	137.4 (45.1)	NS[¥]
			Short base		164.2 (56.7)	183.8 (63.8)	
	Lumbar flexion onset (ms)	↩	***Flat surface***	↩	164.9 (26.8)	108.1 (14.2)	<0.001[¥]
			Short base		210.6 (51.1)	136.1 (18.2)	
	Duration between onset lumbar translation and lumbar flexion (ms)	↩	***Both surfaces***	↩	Longer[$]	Shorter[$]	<0.001
	Onset of first lumbar motion (translation or flexion) (ms)	↩	↩	↩	Later[$]	Sooner[$]	<0.05
	Total excursion of lumbar motion (°)	↩	***Flat surface***	↩	2.36 (1.41)	3.05 (1.45)	NS[¥]
			Short base		2.36 (1.19)	3.25 (1.21)	

Parenthesized values are standard deviations. Bold italic words are linked to presented mean values and levels of significance on the same horizontal level

MAPCH maximum anterior position of contralateral head, *MAPISIPS* maximum anterior position of ipsilateral SIPS, *MCMCSIPS* maximum caudal movement of contralateral SIPS, *MCMISIPS* maximum caudal movement of ipsilateral SIPS, *NA* not applicable, *NR* not reported, *NS* not significant, ↩ the same value/content as above

[$] No mean values reported

[¥] Statistical test was performed with results of flat surface and short base combined

Table 5 Kinetic response to perturbations

Studies	Kinematic parameter	Amplitude normalization	Time window	Perturbation	Plane	Joint	LBP outcome	Control outcome	*p* value pain status
[12]	Mean RMS moment ($M/(m \times g \times l)$)	Weight and leg length	Chair release to 'balance achieved'	20° tilt	Sagittal	***Hip***	0.081 (0.03)	0.083 (0.04)	NS
						Lumbar spine	0.089 (0.03)	0.084 (0.05)	NS
	Maximum moment ($M/(m \times g \times l)$)	↩	↩	↩	↩	***Hip***	0.078 (0.02)	0.087 (0.03)	NS
						Lumbar spine	0.067 (0.02)	0.072 (0.03)	NS
	Mean RMS power: ($P/(m \times g^{1/2} l^{3/2})$)	↩	↩	↩	↩	***Hip***	0.06 (0.03)	0.06 (0.04)	NS
						Lumbar spine	0.010 (0.01)	0.013 (0.01)	NS
	Maximum–minimum power ($P/(m \times g^{1/2} l^{3/2})$)	↩	↩	↩	↩	***Hip***	0.19 (0.11)	0.20 (0.14)	NS
						Lumbar spine	0.04 (0.04)	0.06 (0.05)	NS
[30]	Peak torque (Nm)	Height and weight	25–250 ms after perturbation onset	All directions with sagittal/coronal component	***Sagittal***	Trunk	NR	NR	NS
					Coronal		NR	NR	NS
	Rate of torque development (Nm/s)	Height and weight	Peak-to-peak (minimum to maximum torque)	↩	***Sagittal***	↩	NR	NR	NS
					Coronal		NR	NR	NS
	Peak torque latency, initial trunk response (ms)	Height and weight	25–100 ms after perturbation onset	↩	***Sagittal***	↩	Earlier[$]	Later[$]	0.003
					Coronal		Earlier[$]	Later[$]	<0.05

Parenthesized values are standard deviations. Bold italic words are linked to presented mean values and levels of significance on the same horizontal level

g gravity acceleration (9.81 m/s^2), *l* leg length (m), *m* body mass (kg), *M* moment, *NR* not reported, *NS* not significant, *P* power, ↩ the same value/content as above

[$] No mean values reported

Table 6 Estimated mechanical properties of the trunk

Studies	Estimated property	Order system	Time window	Perturbation	Plane	Joint	LBP outcome	Control outcome	*p* value pain status
[25]	Damping ratio (Nm/(rad/s))	Second order	Chair release to 'balance achieved'	***10° tilt***	Sagittal	Chair	0.2 (0.1)	0.3 (0.1)	NS
				20° tilt			0.3 (0.1)	0.3 (0.1)	NS
	Natural frequency (rad/s)	↩	↩	***10° tilt***	↩	↩	3.5 (0.9)	3.5 (0.9)	NS
				20° tilt			3.5 (0.9)	3.5 (0.9)	NS
[21]	Trunk stiffness (N/mm)	Second order	As long as the load cell measured a tensile force (while the trunk was being pushed)	1 cm anterior and posterior push	Sagittal	L5-S1	NR	NR	NS
	Effective trunk mass (kg)	↩	↩	↩	↩	↩	NR	NR	NS

Abbreviated values are standard deviations. Bold italic words are linked to presented mean values and levels of significance on the same horizontal level

NR not reported, *NS* not significant, ↩ the same value/content as above

anterior position of the ipsilateral PSIS in reaction to the perturbation. In a study in which a weight was dropped in a container held in the hands of standing subjects standing on multiple surfaces, it was found that initiation of lumbar flexion occurred later in LBP patients, without significant differences in the range of motion of the lumbar spine, or the onset of anterior lumbar translation relative to the environment [28]. A study that imposed an anterior push to the trunk reported no significant between group differences in kinematic outcomes [21].

Kinetic response to perturbation

An overview of studies that assessed kinetic outcomes is presented in Table 5. In the two studies that reported the kinetic response to perturbations, subject were perturbed by release of a swing chair [12], or by translation of the standing surface [21]. In the swing chair experiments, no significant between group differences were found in terms of hip and trunk moments and powers. In the standing surface perturbation experiment, the first peak in trunk moment (within 25–100 ms after perturbation) occurred earlier in LBP patients. No differences in maximal trunk moment or the rate of moment development were reported (within 25–250 ms after perturbation).

Estimated mechanical properties of the trunk

An overview of the two studies that assessed estimated mechanical trunk properties is presented in Table 6. Subjects were perturbed in a seated position in both studies [12, 21]. In the experiment in which a swing chair was released, no significant between group differences in trunk damping, and natural frequency of the trunk in the sagittal plane were reported [12]. In an experiment in which the trunk of subjects was pushed in anterior and posterior directions with the pelvis fixed on a chair, no between group differences in sagittal trunk stiffness or effective trunk mass were reported [21]. The LBP subjects in this experiment suffered from 'exercise induced LBP'. After recovery from this LBP the estimated sagittal plane trunk stiffness in this group was significantly higher than in the control group.

Pooling of results

Statistical comparison of outcomes from four blocks (muscle activity amplitude before and after perturbation, muscle activity timing and kinematic amplitude) were presented by three or more studies and hence pooled (Table 7). We found that only the evidence for splinting behaviour in LBP in terms of longer response times of trunk muscles merits further attention. No indications for altered amplitudes of muscle activation, or kinematic responses were found. Between-subject variance was pooled for two blocks of outcomes (muscle activation and kinematics). No indications for variable muscle activation strategies between LBP patients were found (Supplement 3).

Discussion

The aim of this systematic review was to assess whether LBP patients demonstrate signs of splinting by evaluating the anticipation and reactions to unexpected mechanical perturbations in terms of trunk muscle activity, kinetic and kinematic trunk responses and estimated mechanical properties of the trunk. To test if variability may have

Table 7 Within group variability of reported outcomes

Parameter	Studies	In patients with LBP mean outcome is				
		Lower/shorter		NRNS or equal*	Higher/longer	
		$p < 0.05$	NS	NS	NS	$p < 0.05$
Pre-perturbation muscle activity amplitude	[30]	1 (12,5%)	–	7 (87.5%)	–	0 (0%)
	[23]	0 (0%)	0 (0%)	2 (20%)	3 (30%)	5 (50%)
	[16]	0 (0%)	1 (20%)	1 (20%)	3 (60%)	0 (0%)
	[21]	0 (0%)	–	1 (100%)	–	0 (0%)
	[27]	0 (0%)	–	5 (100%)	–	0 (0%)
	Mean¥ (%)	2.5		87.5		10
Muscle activity amplitude	[12]	0 (0%)	–	0 (0%)	–	4 (100%)
	[30]	0 (0%)		21 (87.5)		3 (12,5%)
	[23]	0 (0%)	–	6 (75%)	–	2 (25%)
	[16]	0 (0%)	5 (100%)	0 (0%)	0 (0%)	0 (0%)
	[29]	1 (33%)	2 (67%)	0 (0%)	0 (0%)	0 (0%)
	[17]	7 (70%)	0 (0%)	3 (30%)	0 (0%)	0 (0%)
	[27]	0 (0%)	–	10 (100%)	–	0 (0%)
	[18]	2 (67%)	0 (0%)	0 (0%)	1 (33%)	0 (0%)
	[19]	2 (50%)	0 (0%)	0 (0%)	0 (0%)	2 (50%)
	[20]	0 (0%)	1 (100%)	0 (0%)	0 (0%)	0 (0%)
	Mean¥ (%)	22		59		19
Muscle activity timing	[12]	4 (100%)	–	0 (0%)	–	0 (0%)
	[23]	0 (0%)	–	4 (100%)	–	0 (0%)
	[15, 24]	0 (0%)	1 (25%)	0 (0%)	3 (75%)	0 (0%)
	[16]	0 (0%)	0 (0%)	0 (0%)	3 (60%)	2 (40%)
	[21]	0 (0%)	–	0 (0%)	–	1 (100%)
	[29]	0 (0%)	0 (0%)	0 (0%)	0 (0%)	3 (100%)
	[27]	0 (0%)	–	5 (100%)	–	0 (0%)
	[18]	0 (0%)	0 (0%)	0 (0%)	1 (17%)	5 (83%)
	[13]	0 (0%)	0 (0%)	0 (0%)	0 (0%)	3 (100%)
	[14]	0 (0%)	0 (0%)	0 (0%)	2 (50%)	2 (50%)
	Mean¥ (%)	10		43		47
Kinematics amplitude	[12, 25]	2 (50%)	2 (50%)	0 (0%)	0 (0%)	0 (0%)
	[26]	0 (0%)	2 (50%)	0 (0%)	0 (0%)	2 (50%)
	[21]	0 (0%)	–	1 (100%)	–	0 (0%)
	[28]	0 (0%)	2 (100%)	0 (0%)	0 (0%)	0 (0%)
	Mean¥ (%)	12.5		75		12.5

The table displays the number of times reported outcomes were significantly higher, not significantly different or significantly lower in LBP subjects compared to healthy controls. If outcomes were not significantly different and mean values were provided the table shows if the reported means in the LBP group were decreased/lower or increased/higher compared to the control group

NRNS If mean values are not reported and between group differences were not significant, – Cell empty because mean values were not reported, only statistical significance of between group differences

*Reported mean values were identical between groups

¥ Calculated by averaging the percentage of outcomes in each study over studies

masked group differences within the LBP population, we evaluated within group variances as well. No sign for increased variance within the LBP group was found. We found evidence in line with splinting behaviour in LBP in terms of a longer response time of muscle activation, which merits further attention. No signs of splinting behaviour were found for any of the other outcome measures.

Longer response time of trunk muscle activation may occur as a result of splinting in response to LBP, but they have also been identified as a risk factor for developing

LBP [31]. It was found that college athletes who showed longer response times of relaxation of trunk muscles in a sudden release experiment were at higher risk of developing LBP. Increased latencies of trunk muscles may require higher reactive muscle forces in reaction to external perturbations [32], which could lead to injury and LBP. It could be that the longer response times present before getting LBP (not explained by splinting behaviour) [31] remain present after LBP develops. In addition, the interpretation of increased response times of muscle activation in LBP requires some caution. First of all, these response times should not be interpreted as reflex delays (a term used by many of the included papers in this review). Response times are dependent on both reflex delays and the initial conditions of the trunk. If the initial resistance of the trunk to a perturbation is increased by a higher trunk mass, trunk stiffness or damping, the acceleration of the trunk will be lower, which may well result in longer response times for a given reflex delay, due to later detection by the sensory system. Second, it is possible that longer response times of trunk muscles in LBP patients are the result of a bias in data analysis. In most studies in which response times of trunk muscles were evaluated, the first muscular response was defined as the instant at which an EMG signal exceeded a predetermined number of standard deviations above baseline activity. Hence, the reported response time is influenced by both the mean and within-subject variance of baseline muscle activity. Although mean baseline activity was reported in most studies, none of the included studies reported the within-subject variability of this baseline activity. Increased variability of trunk muscle activity has been reported in LBP during gait [33], but, to the best of our knowledge, has not been evaluated in this population during static tasks. If mean baseline activity and the muscular response to a perturbation are identical between subjects, one would expect to find longer latencies of muscle activation in subjects with higher baseline variability of muscle activity.

In all of the four blocks of outcomes that were pooled, e.g., pre-perturbation muscle activity, timing and amplitude of muscle activity and amplitude of kinematics, conflicting significant between group differences were reported by at least two studies per block. The two most likely explanations for these differences are the usage of different experimental setups and the methods for data analyses. The study that found a significantly decreased pre-perturbation muscle activity normalized EMG signals to the maximum value of that muscle measured over all trials [30] whereas the studies that reported increased amplitudes of back muscles both utilized maximally voluntary contractions and reference contractions to normalize the data. The one study that found deviating significant results when compared to the other studies in muscle activation amplitude and kinematic amplitudes was the only one in which subjects had to recover from a perturbation on an unstable seat [12]. It is likely that such a condition requires a different motor control strategy, because stiffening of the spine will not result in stabilization of the seat.

It is possible that signs of splinting were present in the investigated LBP cohorts, but overlooked for at least two reasons. First of all, the performed analyses of the muscle responses, kinematics and kinetics could be sub-optimal. Summarizing a one-dimensional, i.e., time varying, reaction to a perturbation with a discrete value, e.g., maximal amplitude, might be an oversimplification of the data. Not only does this increase the chance of type I errors [34], it also has negative consequences on the comparability of results between studies. All studies evaluated the reactions to perturbations over one or more arbitrarily chosen time-window(s) and reported discrete outcomes within these windows. The reaction to a perturbation within a time window can be quite complex. For instance, the EMG signal can contain multiple peaks, e.g., monosynaptic and polysynaptic reflexes and voluntary responses. In that case, discrete outcomes are difficult to interpret. For the same reason, apparently conflicting results between studies could be the consequence of different adopted time-windows. One study that assessed the muscular response over two time windows, i.e., 40–120 ms and 40–250 ms after perturbation onset, reported a significant decrease in abdominal and back muscle amplitude in LBP patients over the first time window and a significant increase over the second [22], which underpins that the comparability of studies that applied different time-windows is limited.

Secondly, the adopted models to estimate the mechanical properties of the trunk might be over-simplified. The effect of perturbations on the kinematics of the trunk depends both on intrinsic and reflexive components [8]. In the two studies that estimated mechanical trunk properties [21, 25] only one lumped value (i.e., comprising information on both the intrinsic and reflexive component) of each parameter was calculated. To determine whether splinting is present in LBP patients, the intrinsic stiffness of the trunk should be isolated, which was not done in the included studies.

As a result of the variation in experimental setups and analysis methods, evidence for splinting behaviour remains inconclusive. Increased estimated spinal stiffness in LBP was found in a study among patients with recurrent low back pain (in a pain free episode and therefore not included in this review) [35]. A later study reported a significant positive correlation between estimated spinal stiffness and fear of movement in LBP [36]. This study utilized a control group from the aforementioned experiment [35] that did not use the same perturbation force. Therefore, this study was also not included in this review.

Several recommendations for future research on postural control of LBP patients can be made. First of all, it is recommended to study the trunk in isolation, with a restrained pelvis and perturbations imposed directly to the trunk [37]. This prevents that other segments of the body influence the results and makes interpretation of the data more straightforward. Second, instead of using a lumped model to predict mechanical properties of the trunk, it is recommended to estimate both intrinsic and reflexive components using system identification [38]. Third, to statistically compare one-dimensional data, techniques should be used that are designed for time series analysis like wavelet-based functional ANOVA's [39] and one-dimensional statistical parametric mapping [40]. Finally, when reporting EMG results, measurements that are used to normalize the signal, or to calculate a threshold, should be reported to give more insight in possible biases, e.g., pain-related inhibition during MVC, increased co-contraction during a reference contraction and/or thicker subcutaneous fat in patients. For example, the EMG-amplitude and generated torque during an MVC used for normalization should be reported and the mean and variability of baseline EMG-signal used to determine response time to a perturbation as well.

We conclude that there is currently no convincing evidence for the presence of splinting behaviour in LBP patients, because we found no indications for splinting in terms of kinetic and kinematic responses to perturbation or the derived mechanical properties of the trunk. The indication of delayed onset of muscle activation in reaction to perturbations deserves further attention. Standardized experimental protocols and more advanced data analyses should be utilized in future research to provide conclusive evidence for the splinting hypothesis in low back pain.

Compliance with ethical standards

Conflict of interest None of the authors has any potential conflict of interest.

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